ENDOTRACHEAL INSUFFLATION WITH OXYGEN IN THE TREATMENT OF ASPHYXIA NEONATORUM

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The present state of our knowledge of the oxygen and carbon dioxide economy of the newborn gives every support to the belief that the chief therapeutic need in severe asphyxia neonatorum is for oxygen, and that the particular means by which this is administered is of less importance, provided it is efficient. The assessment of any one method of resuscitation is of course hard to establish. In the first place there is the difficulty of finding a sufficiently large number treated according to one routine; in the second place there is the impossibility of obtaining an adequate control series either from another hospital or in the same hospital if the routine is felt to be of value.

For these reasons we are recording briefly the long-term results of treating 66 severely asphyxiated infants by endotracheal insufflation with oxygen. These cases occurred from 1949 to 1951, during which time there were 5,458 deliveries.

Methods

The methods employed have already been reported in full by one of us (Roberts, 1949), and may briefly be restated as follows. Whenever possible a member of the medical staff was present to care for the infant when asphyxia was anticipated. In addition there was a resident paediatric registrar on call, who, with the anaesthetists and obstetricians, was versed in the technique of endotracheal insufflation. In all babies the mucus was aspirated from the pharynx immediately after birth; those who did not breathe immediately were then placed head down in a Roberts-Talley infant oxygen rocker. Where respiration had not started by three minutes after birth, a small metal airway was inserted and the infant rocked. The group reported here consists of those who after a further three minutes' rocking—that is, six minutes altogether from birth—still showed no response. The pharynx was then aspirated under direct vision, using an infant laryngoscope, and a No. 00 Magill endotracheal tube inserted. This was attached to a Y-shaped metal connexion which enabled the operator to apply oxygen at intermittent pressures of about 20 cm. of water by occluding the open end for periods of approximately three seconds between 10 and 15 times a minute. The optimum rate of flow for this purpose was 1½ litres a minute. It was found to facilitate the insertion of the tube through the glottic opening if it was threaded with 10-amp. copper fuse wire for about seven-eighths of its length. This allowed the curve of the tube to be maintained.

Insufflation was maintained for as short a time as possible and the tube was withdrawn as soon as respiration began. This interval varied from 3 to 40 minutes, but was usually about seven minutes.

Results

Altogether 66 infants were intubated; of these 14 died. The causes of death are given in the accompanying Table. The remaining 52 infants made an uninterrupted recovery,

Table Showing Causes of Death

Case	Maternal Complication	Delivery	Birth Weight lb. oz.	Time of Death	Post-mortem Findings
<i>A. Cases Other Than Asphyxia</i>					
1	Pre-eclamptic toxæmia	1st twin, caesarean	6 1	9 mths	Pneumonia
2	Infective hepatitis; fibroids	Caesarean	3 12	3 wks	Extensive hepatic necrosis
3	Pre-eclamptic toxæmia	Spontaneous vertex	4 4	31 days	Gastro-enteritis
<i>B. Asphyxia a Contributory Cause</i>					
4	Nil	Difficult vertex	9 4	1½ hrs.	Congenital urethral valve; gross ascites; hydro-ureter; hydro-nephrosis; atelectasis
5	Carcinoma	Therapeutic hysterotomy at 26 weeks	2 13	5 hrs.	Extensive atelectasis
11	Nil	Vertex; cord round neck	6 8	15½ hrs.	Extensive pneumonia
13	"	Breech	5 0	3 days	Capillary haemorrhage in right parietal region; minimal atelectasis
14	"	2nd twin, vertex; prolapsed cord	4 0	4 "	No necropsy
<i>C. Asphyxia the Principal Cause of Death</i>					
6	Premature rupture membranes	Spontaneous vertex	2 5	1 hr.	Extensive atelectasis
7	Severe pre-eclamptic toxæmia	Caesarean	4 0	2 hrs.	Partial atelectasis; some subpleural emphysema
8	Nil	Forceps for foetal distress	7 2	5 "	Moderate atelectasis; vascular engorgement in thalamic region
9	Antepartum haemorrhage due to placenta praevia	Caesarean	3 6	8 "	Aspiration of amniotic contents; atelectasis
10	W.R. positive	Breech, 2nd twin	4 10	16 "	Widespread aspiration of amniotic fluid and pulmonary interstitial haemorrhage
12	Severe pre-eclamptic toxæmia, uterus bicornis	Caesarean	4 7	21 "	Pulmonary oedema from extensive aspiration; hyaline membranes

although two showed transient signs of cerebral irritation in the first 10 days of life—one mild, and one severe enough to be diagnosed as a cerebral haemorrhage. As it is not possible in the immediate post-natal period to assess either the duration of intrauterine anoxia or the possible permanent damage to the central nervous system, we decided to institute a long-term follow-up of this group. It seemed just possible that by waiting as long as six minutes we might be exposing these infants unduly to the risk of subsequent mental debility.

The results of this part of the investigation are as follows. It was found impossible to trace six infants. Three infants were below 3 months of age and were not developmentally tested. None of these had shown signs of cerebral irritation or locomotor disturbance during their stay in hospital. The remaining 43 infants were examined clinically, and their degree of development was tested by one of us (D. O'B.), using the technique of Gesell and Amatruda (1949). All of these children appeared normal in every way, except two who from shyness or unfamiliarity with the surroundings co-operated indifferently at the first examination. These two were subsequently re-examined by an independent observer and adjudged normal.

Discussion

Although in this series we feel that our intervention undoubtedly saved the lives of a number of profoundly asphyxiated infants, we are not in a position to prove it. However, where recovery occurred it has been uncomplicated by any permanent cerebral damage. Similar results have been obtained in another study (Usdin and Weil, 1952),

although only 9 of their 40 reported cases would have been included in our group. Of the 14 deaths three were from causes other than asphyxia, and two more—a therapeutic abortion and another with a gross congenital abnormality—would very probably have occurred whatever methods were employed. Of the remaining nine fatal cases, seven responded initially to insufflation, although they died later after periods ranging from five hours to four days with varying degrees of atelectasis and evidence of amniotic aspiration. Of the two who failed to respond, one was a very small premature infant and the other was the only case to show a mild degree of subpleural emphysema.

We have in addition tried to obtain information on precisely how insufflation may modify the physical state of the lungs. The rapid oxygenation of the asphyxiated, as shown by skin colour, the movement of the chest wall, and the showers of fine crepitations on auscultation at each insufflation, suggested that the alveoli themselves were being expanded. Unfortunately, we were not able to obtain any radiographic records in the labour wards; a number of photographs, however, were obtained very shortly after death. Two of these are shown (Figs. 1 and 2), and they suggest that a pressure of +20 cm. of saline is sufficient to produce a considerable degree of alveolar expansion whilst it is exerted.

FIG. 1.—Case H. Birth weight 4 lb. (1.8 kg.). Control picture showing extensive atelectasis.

FIG. 2.—Same infant as in Fig. 1, showing alveolar expansion with a sustained intratracheal pressure of +20 cm. of saline.

In addition we have practised this technique in eight very small premature infants who appeared to be dying as a result of atelectasis, in one case continuing for eight hours. In all these there was a transient recovery, as evinced by a rise in pulse rate and improved colour. Life never seemed to be prolonged more than a few hours, and the manipula-

tions of inserting the tube and changing it when blocked with mucus were badly tolerated. Extensive atelectasis without emphysema was revealed in all cases at necropsy. This information, together with that from the post-mortem reports of babies dying of asphyxia, suggests that endotracheal insufflation with the pressures we have used does not to any perceptible degree facilitate the permanent expansion of the atelectatic lung in the newborn, although it may do so in some dead or stillborn infants, especially if full-term. A satisfactory degree of oxygenation of the blood and tissues can, however, be produced almost immediately.

Finally, we would stress the desirability of an established routine in the treatment of asphyxiated infants. For convenience this may be primarily but not exclusively the responsibility of the anaesthetist, the obstetrician, or the paediatrician. To ensure prompt action in the unexpected emergency all should be proficient in the technique employed.

Conclusions

The administration of oxygen under intermittent positive pressure of up to +20 cm. of water by endotracheal tube is, in experienced hands, a safe, quick, simple, and efficient treatment for severe cases of asphyxia neonatorum.

This procedure, though momentarily expanding the alveoli, does not appear permanently to modify the physical state of the unexpanded newborn lung.

We would like to thank Professor J. C. McClure Browne and Professor A. A. Moncrieff for permission to carry out these investigations on infants under their care, and also Dr. C. Upjohn and Dr. M. W. Arthurton, who intubated many of these babies and assisted in keeping the records. Dr. B. W. Powell, Assistant to the Professor of Child Health, kindly reassessed the developmental age of two infants.

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THE RESPIRATORY EFFECTS OF SALICYLATE

BY

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There is an extensive literature on the effect of salicylates on respiration and on the acid-base balance of the blood. Most observers agree that salicylate causes hyperventilation and a reduction in the carbon dioxide content of the blood. There has been great controversy, with many conflicting reports on the interpretation of these findings: whether salicylate causes a primary respiratory alkalosis or a metabolic acidosis with secondary hyperventilation (Johnson, 1930; Morris and Graham, 1931; Rapoport and Guest, 1945; Reid, Watson, and Sproull, 1950).

It was decided to investigate the respiratory effects of orally and intravenously administered salicylate in the human subject. For this purpose a closed-circuit Knipping type spirometer was used. This apparatus measures oxygen consumption, carbon dioxide output, and depth and rate of respiration. Most previous observations on the effect of salicylates on the respiratory movements in the human subject have been made